## American Medical Association

Physicians dedicated to the health of America



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Comments to Docket Number H-122 American Medical Association 506

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Recent reports in the press and elsewhere have made much of a new report by the Congressional Research Service (CRS) on environmental tobacco smoke (attached).

A thorough reading of the CRS report on the health effects of environmental tobacco smoke (ETS) leads to very different conclusions about ETS than those stated by tobacco industry apologists. The CRS report makes two kinds of calculations for the potential lung cancer risks from ETS exposure, based on either a "no-threshold model" that implies no safe lower limits for ETS exposure, or a "threshold" calculation based on an assumption that only persons at the highest levels of ETS exposure are at risk. The authors are careful to point out that the "threshold" method "is a hypothetical example and does not mean that any lung cancer which might result from ETS exposure would actually exhibit a threshold dose response behavior relationship." The basis for these calculations are two studies that, according to the CRS report, "provide some indication of the possibility of a threshold," hardly iron-clad data to conclude that the CRS report "makes hash" out of the risks of ETS exposure, as claimed by some in the press.

Using the "no-threshold" model, the basis for the original Environmental Protection Agency (EPA) report, the CRS arrives at risk estimates almost identical to those of the EPA: about 2800 lung cancer deaths per year among nonsmokers from ETS, compared with EPA's estimate of 3300. The "threshold" method comes to about 440 deaths per year. The CRS authors admit that " even if the threshold model were approximately correct, public health officials may still chose (sic) to use a model closer to the non-threshold approach in order to ensure that all populations are protected."

The CRS report also states that "If one accepts that there is a causal link between residential

The CRS notes that many potential confounding elements exist in studies of lung cancer and ETS, but concludes that attempts to determine whether dietary vitamin and fruit intake, saturated fat intake, and other variables appear to be-"inconclusive."

It appears, based on these statements in the CRS report, that the document in fact makes the case that ETS poses a serious risk regarding lung cancer. The CRS report also states that there exists a "potentially large public health impact of ETS on heart disease" and calls for a "comprehensive assessment and additional research program" on this issue.

Comments by Gori delivered to the Docket August 31, 1995, state (p. 5) that "the contribution [of ETS] to IAQ is insignificant... compared to other factors such as building materials... and outdoor air contributions." Outdoor air contaminants, for example, account for a vanishingly small number of cancer deaths per year. The following table from Repace and Lowery's article, "Risk assessment methodologies for passive smoking-induced lung cancer" (Risk Analysis 10 (1): 27-37, 1990) puts these into perspective:

Asbestos 15 deaths/yr
vinyl chloride <27 deaths/yr
Airborne radionuclides 17 deaths/yr
Coke-oven emissions <15 deaths/yr
Benzene <8 deaths/yr
Arsenic <5 deaths/yr

His statement concerning "other factors" in IAQ as having significant mortality as compared with ETS estimates is unsubstantiated. ETS itself can be a major source of hazardous chemicals in the indoor environment. For example, ETS can contribute as much as 250 ppb of formaldehyde and 75 ppb acrolein to the indoor atmosphere. (Leikauf GD. Formaldehyde and other aldehydes. In: Lipmann M (ed). Environmental Toxicants. Van Nostrand and Reinjold, NY 1992.) A report from a symposium partially sponsored by the tobacco industry also finds that "cigarette smoke contributes to building sickness" (Robertson AS, Sherwood-Burge P, et al. The relationship between passive cigarette smoke and symptoms of "building sickness." In: Indoor and Ambient Air Quality. Selper Publications, London 1988.)

Information on ETS particle emissions and toxic volatile compounds has been compiled at the Lawrence Berleley Laboratories at the University of California, and are attached.

Gori uses the report of Congressional economists, Gravelle and Zimmerman, authors of the Congressional Research Service report as an authoritative source for much of his comments. These individuals are not health or epidemiology specialists, but economists who looked at cigarette taxes in their report, and only examined the science of tobacco as it applies to health by default in analyzing, for economic purposes, mortality data. The Centers for Disease Control authored a rebuttal to the CRS report (attached), with which we concur.

The AMA, the American Psychiatric Association, the American Society of Addiction Medicine, the National Institutes of Drug Abuse, the American Psychological Association, and many other groups have all studied nicotine dependence, and conclude that there is an addiction to nicotine from which physiologic withdrawal occurs. Indeed, nicotine dependence and its withdrawal are listed in the definitive "Diagnostic and Statistical Manual, fourth edition (DSM-IV) of the American Psychiatric Association (excerpts attached).

Gori is no expert in these matters. But if no addiction is present, as Gori argues, smokers should not object to the proposed restrictions on indoor smoking, since they are only doing so for the "taste" of nicotine, according to the industry, not its pharmacologic effects on the brain. The tobacco industry, of course, objects to the proposed OSHA tobacco proposals because smokers consume less cigarettes per day in restricted work settings, and it costs the industry money.

Gori seems not to think that the studies by Penn and others on ETS and athetotic plaque development in experimental animals are meaningful. While the experiments in question might have been done more elegantly, as described by Gori, the flaws are not fatal to the outcome, and it is interesting that all the experiments come to similar conclusions despite differing design. He also fails to note that human subjects exposed to ETS, for instance, experience angina and vasoconstriction of coronary stenoses (Brown RE, Nahser, PJ et al Vasoconstriction of coronary stenoses from exposure to environmental tobacco smoke. J Am Coll Cardiol 1994: 23(2 sup)107A.) It is also a hazard to persons with known coronary infarctions (Leone A. Passive smoking in infarcted patients: role of indoor exposure. In Indoor and Ambient Air Quality. Selper Publications, London 1988.)

Comments by Professor Jeffrey R. Idle dated May 30, 1995 contain an assertion that ingesting vegetables that naturally contain nicotine (eg potatoes, tomatoes, etc.) could significantly affect urinary excretion of cotinine, thereby falsely influencing exposure measurements to ETS. Letters to the editor of the British Medical Journal on the subject are attached, pointing out that dietary nicotine is an insignificant contributor to urinary cotinine.

The American Medical Association maintains the position that it is unacceptable to continue the exposure of workers to tobacco smoke in the environment. We also urge that the section of the proposed regulations on ETS, given that is would be inexpensive to put in place by source elimination, be separated from the other sections of the regulations and put in place immediately. The State of Maryland has promulgated regulatory actions that have many

similarities with the OSHA regulations as they relate to ETS, and personal communications with state government and public health officials relates little difficulty in their implementation.

Sincerely,

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American Medical Association